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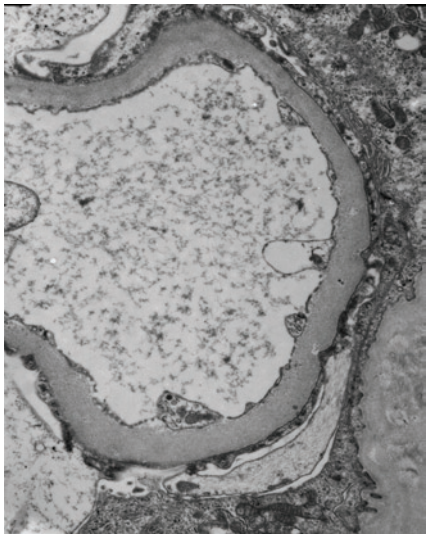
Foot process effacement in FSGS



Electron microscopy is an important tool in the analysis of the structure and function of the glomerular filtration barrier. The foot processes of healthy podocytes are seen to be separated from each other. But when any disease causes proteinuria, these processes become 'effaced' and thus appear to fuse with each other. In this issue, Deegens *et al.* provide a detailed measurement of each foot process in normal kidneys compared with those in diseased kidneys. The authors found that the width of each foot process in normal kidneys was 562 nm, whereas the foot processes of patients with minimal-change nephrotic syndrome were much wider, an average of 1725 nm. Analysis of patients with primary and secondary focal segmental glomerulosclerosis (FSGS) showed that the foot processes of patients with idiopathic FSGS were 3236 nm, whereas those of patients with secondary FSGS were much thinner, 1098 nm. These findings did not correlate with the magnitude of patients' proteinuria; patients with foot processes of 1500 nm or wider had idiopathic FSGS. This new criterion should help differentiate between idiopathic and secondary FSGS, two diseases that are difficult to distinguish by other morphologic criteria. The question of the basis of this difference in width is intriguing and should stimulate new studies on the structure of the foot process in kidney disease. **See page 1568.**

Vascular calcification of vascular access: a predictor of overall mortality

Increasing evidence exists that vascular calcification is one of the more severe consequences of end-stage renal disease. Calcification in arteriovenous shunts predicts short-time survival of the shunt itself but may or may not have any additional predictive value. In a new study, Schlieper *et al.* evaluated the shunts of more than 200 patients on hemodialysis using radiographic means. The shunts of almost one-quarter of these patients had vascular calcification. The authors then evaluated a number of parameters, including the length of dialysis, a variety of serum laboratory findings, and the presence of diabetes. They found that male gender, diabetes mellitus, and a longer history of dialysis were independent predictors of vascular calcification. Further, vascular calcification independently predicted mortality. Surprisingly, the serum factors were not independent predictors of calcification. **See page 1582.**

Vitamin D suppresses renin

Recent studies have suggested that vitamin D regulates renin expression. Some of these studies were performed in vitamin D receptor knockout mice, while others used *in vitro* experiments. Now, Kong *et al.* provide *in vivo* studies of this process. They generated transgenic mice that express the human vitamin D receptor driven by the renin promoter. When the vitamin D nuclear receptor was overexpressed in the juxtaglomerular cells, renal renin mRNA levels as well as plasma renin activity were reduced. No significant change in the blood pressure, plasma calcium, or parathyroid hormone occurred. The authors also overexpressed the human receptor in the juxtaglomerular apparatus of vitamin D receptor knockout mice. Again, they found reduction in renin. These results show that vitamin D directly regulates renin expression independent of parathyroid hormone and calcium. **See page 1577.**